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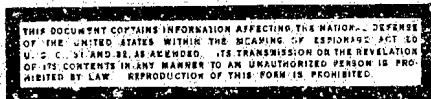
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CASES OF ACUTE POISONING BY SO-CALLED
EXPLOSION CASES IN Leningrad IN 1943

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(The following paper was read at the 19th Leningrad City Conference on Problems of Toxicology and Sanitary-Chemical Defense, 20 July 1944.)

During World War II, Leningrad, while under enemy blockade, was frequently subjected to intense artillery fire. The authors of this paper had the opportunity of personally examining four bombardment victims indicating peculiar intoxication phenomena. Notes of these observations form the material for this paper.

On 17 July 1943, four men, victims of the artillery bombardment of the city, were brought to the receiving ward of one of the therapeutic institutions. These cases had taken refuge in a basement at the beginning of the bombardment. As the bombardment intensified and shells fell in the area they retreated for safety to a nook where there were no windows. However, a shell hit the cellar and exploded right at the entrance, showering the men with fragments of brick and lumber. Since bombardment of the area was still in progress, the men remained in the basement. However, the cellar became so full of smoke that the refugees actually thought a fire had broken out in the building. They were compelled to remain for about another hour under these conditions, after which the shelling decreased somewhat, but the situation became more difficult for the refugees because of smoke. Victim S. began to vomit and soon afterward lost consciousness.

With the help of two who arrived at the scene, as well as that of victims N, A, and G, the unconscious man was brought to the receiving ward. He vomited several times during his stay in the receiving ward and remained unconscious. In view of the fact that efforts to restore consciousness were unsuccessful, even with artificial respiration and the administration of oxygen, one of the authors of this paper was summoned by the attending physician an hour after the victim had been admitted to the receiving ward. Examination revealed hyperemia of the face

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and mucous membranes, pulse 60-65 with good rhythmic beat, respiration 20-22, and somewhat labored, dull heart tones.

Approximately 1½ hours after Victim S was admitted to the therapeutic institution, a physical examination was carried out and his blood was tested for carboxyhemoglobin. Results were as follows: hemoglobin 76 percent, red count 3,700,000, white count 5,400, and carboxyhemoglobin positive (qualitative reaction according to Goppe-Zeyler, a modification of Sal'kovskiy method). Victim S regained consciousness after inhaling carbogen [sic] for 40 minutes. The victim complained of persistent headache, giddiness, and nausea. However, he categorically refused to be transferred to a clinic because he had to attend to urgent business and, 3 hours later, having declared that he was feeling all right, insisted on being discharged. A second blood test for carboxyhemoglobin after the patient had regained consciousness gave a negative result.

Victim G, age 31, came to the receiving ward complaining of headache, giddiness, nausea, and back ache sustained when he was flung against the basement wall by the concussion of the shell burst. An objective examination by the attending physician revealed a moderate hyperemia of the face and mucous membranes. No internal abnormalities were indicated. Approximately an hour after being admitted to the receiving ward the victim declared that he felt all right and was sent home. The attending physician diagnosed Victim G as a case having a slight contusion.

Victim A, age 28, came to the receiving ward without assistance and even helped in bringing his unconscious comrade. When questioned about his condition by the attending physician, Victim A complained of headache and, especially, of pain in the sacral region, attributing this mainly to being flung against the wall by the concussion. Moreover, he complained of giddiness, the feeling of intoxication, and nausea. Slight vomiting took place twice during the first hour of his stay in the receiving ward. Objectively no abnormal developments were observed by the attending physician. Pulse rate was 85, with a good rhythmic beat. The victim was placed on a cot in the receiving ward and fell asleep comparatively quickly. He awoke approximately 3 hours later and his condition showed a marked change for the worse: the headache was considerably worse; he complained of a feeling of compression in the chest, acute laborious respiration, and increasing myalgic asthenia. Cough began to disturb the patient considerably. The body temperature at this time was 38.2 degrees, pulse 100 of good, rhythmic beat, and respiration 28. The face became more and more cyanotic. The patient was transferred from the receiving ward to the station hospital, where he was examined by one of the authors of this article approximately 4 hours after his arrival at the receiving ward.

Victim A repeated his complaints mentioned above, saying that it was getting harder to breathe all the time. His pulse was 110, respiration 36. Pronounced cyanosis of lips and ears. Dipsoesis. The cough became stronger and stronger, and soon this was accompanied by expectoration of sputum mixed with blood. Auscultatory percussion disclosed a hollow sound about the 8th, 10th and 12th costal region. Diaphragm almost motionless, heart covered by the pulmonary edge, tones dullish. Auscultation revealed copious dry crepitations in the superior region and nonresonant moist crepitations in the inferoposterior region. Blood pressure 115/70.

Blood examination: hemoglobin 90 percent, red count, 5 million, white count 11,000, lymphocyte 15 percent, neutrophile 79 percent, transitional forms 3 percent. Carboxyhemoglobin test proved negative.

Urinalysis: specific gravity 1.018, isolated red blood cells in centrifuged specimen, traces of albumen.

Three hundred cubic centimeters of blood were drawn by venipuncture. Administration of oxygen (prolonged). Intravenous injection of 30 cc of a 40 percent solution of glucose, caffeine subcutaneously.

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Examination of blood 3 hours later (7 hours after the poisoning): hemoglobin 90 percent, red count 4,800,000, white count 10,000, lymphocyte 10 percent, neutrophile 82 percent, transitional forms 4 percent, eosinophile 4 percent.

There was no particular change in the general condition of the patient. Pulse 100-120 of good rhythmic beat, respiration 28-40, body temperature 38-38.5 degrees. Strongly disturbed by asphyxia and cough accompanied by the expectoration of foamy, yellow, sputum, mixed with blood. Cyanosis persisted. Auscultation revealed considerably more moist, medium and small bubblelike crepitation in both lungs.

Carried out a second venipuncture; let out another 300 cc of blood.

On the following day the patient felt considerably better. Body temperature 37.2 degrees, pulse 80 of rhythmic beat, normal respiration of 24. Blood: hemoglobin 85 percent, red count 4,500,000, white count 8,000, lymphocyte 15 percent, neutrophile 82 percent, transitional forms 2 percent, eosinophile 1 percent. Considerable dry crepitations during auscultation, some moist crepitations were indicated in the inferior regions of both lungs.

The patient's condition continued to improve daily and the following data were obtained on the day before his discharge from the therapeutic institutions on 25 July 1943 (ninth day after the poisoning): body temperature 36.7 degrees, pulse 70, respiration 18; blood -- hemoglobin 80 percent, red count 4,200,000, white count 7,000, lymphocyte 29 percent, neutrophile 62 percent, transitional forms 4 percent, eosinophile 5 percent. Heart tones normal. Lungs within normal limits. Urinalysis revealed only trace of albumen.

It was suggested to the patient that he report for ambulatory observation a week later, but he did not come.

Thus, it can be stated that this patient had a typical toxic emphysema of the lungs, moderately expressed, which pursued a favorable course during the observation period.

Victim N, age 30, came to the receiving ward and complained only of headache and nausea, both of which passed after an hour. Since Victim N declared that he was feeling well and insisted on leaving to attend to urgent business, he was discharged by the attending physician.

On the following day, 18 July, 12 hours after leaving the receiving ward and approximately 13½ hours after the incident, Victim N was brought back to the therapeutic institution in very bad condition.

During the 4-5 hours after leaving the receiving ward he had to go about the town a great deal on urgent business during the continuous bombardment. He felt, according to his own words, "not too good," mainly because of a renewed headache, which he ascribed to excitement. When he returned home he felt so bad (headache and myalgic asthenia), that he decided to lie down without a bite to eat, although he had eaten nothing since morning. At night, approximately 10-11 hours after the shell burst, he woke up, covered with perspiration due to a stifling sensation and pain in his chest. While waiting for dawn, and not wishing to disturb his sleeping comrades, he dozed again. He evidently awoke 1½-2 hours later due to the intensification of the above symptom; this was accompanied by a strong nausea, and he vomited twice. His condition was very serious.

When Victim N came to the therapeutic institution he complained of an acute irritating cough, thirst, and a feeling of compression in the chest, accompanied by signs of pronounced stifling, headache, considerable myalgic asthenia, nausea with inclination to vomit, especially when changing the position of the body.

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There was nothing unusual in his anamnesis. He smokes, and has overindulged in alcohol in recent years.

An objective examination revealed that the patient was stout, with well developed muscles. Hyperemia of face and mucous membranes. Lips, tongue, fingers, and tip of nose were cyanosed. Body temperature 38.3 degrees. Respiration 38-39. Pulse 86-94, of weak, rhythmic beat. Blood pressure 110/70. Hollow sound in the lungs and parietal peritoneum. Auscultation revealed moist heart and small-bubble crepitations in the whole pulmonary region, particularly in the right side. Heart was obscured by the lungs; heart tones dull. Copious, foamy sputum, colored yellow with a clear admixture of blood. Tongue dry and coated. The abdomen was distended and painful during palpitation in the right subcostal region, and the liver proved negative.

Emphysematous syndrome increased during the next 8 hours. Respiration became more labored and increased in rate, reaching 58; the cough was accompanied by the expectoration of bloody sputum, and became more intensified. During auscultation, considerable moist crepitations of varied degrees could be heard, particularly along the right lung. Cyanosis became more clearly defined. Sometimes the face took on an ash-gray tinge.

The following was the result of examination of blood during the first 24 hours of Victim's stay in the station ward: hemoglobin 105-115 percent, red count 4,700,000-5,800,000, white count 16,000-21,500, lymphocyte 12-15 percent, neutrophile 82-79 percent, transitional forms 3-2 percent, eosinophile 3-1 percent.

Urinalysis specific gravity 1.023, trace of albumen; leucocytes, 3 - 4 per microscopic field; erythrocytes, isolated ones in centrifuged specimen; hyaline casts, 2 - 3.

Phlebotomy was carried out on the patient (300 cc) and moist oxygen was administered over a prolonged period. Thirty cubic centimeters of a 40-percent solution of glucose were injected intravenously and caffeine was also injected.

On the following day (19 July) body temperature was 38.4-39 degrees. There was no noticeable change in the patient's general condition. Pulse 104-110, of satisfactory, rhythmic, beat; respiration 42-50; blood pressure 107/70. Considerable cyanosis during auscultation. Copious nonresonant moist crepitations of various calibers. Blood: hemoglobin 100-115 percent, red count 4,000,000-5,600,000, white count 17,000-19,000, lymphocyte 10-16 percent, neutrophile 85-78 percent, transitional forms 4-5 percent, eosinophile 1 percent.

Phlebotomy was carried out again (300 cc). Thirty cubic centimeters of a 40-percent solution of glucose were again injected; oxygen and an antemetic was given.

On the following day (20 July) the patient felt considerably better; the cough had decreased, and during auscultation, slight, moist crepitations were considerably less. Heart tones dullish, pulse 85-90, of satisfactory rhythmic beat. Blood: hemoglobin 90 percent, red count 4,600,000, white count 12,500, lymphocyte 20 percent, neutrophile 75 percent, transitional forms 3 percent, eosinophile 2 percent.

The condition continued to improve during the subsequent days, and on the day of his discharge (27 July) his condition was as follows: body temperature 36.7 degrees, pulse 78 (of good rhythmic beat), respiration 20, lungs clear, except for some dull tones in the cardiovascular region. Blood: hemoglobin 82 percent, red count 4,200,000, white count 8,200, lymphocyte 24 percent, neutrophile 72 percent, transitional form 1 percent, eosinophile 2 percent. Urinalysis: specific gravity 1.012; leucocytes, isolated in centrifuged specimen (no albumen, erythrocytes or casts observed).

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It was suggested to all the patients that they report for another examination; however, none of them did. Due to circumstances beyond the control of the authors of this paper, it was not possible to learn what had happened to the patients. Thus, our observations are confined to the data given.

A comparison of the clinical picture of the described diseases with the circumstances under which they occurred leads us to conclude that we are dealing with intoxication by so-called explosion gases.

Poisoning by explosion gases has been described several times in the literature on military toxicology (Edere and Isten) under the title of powder disease, observed mainly during naval and fortress battles, and more rarely under field-warfare conditions in shelters and recently formed shell craters. Similar poisonings have been described during blasting in coal mines (Kavalerov and Ardashev, Aynbinder, and others) and in tunnels (Metsatun'yan).

The composition of the gases formed during explosions can vary considerably, depending on the composition of the explosive and the completeness of its combustion upon explosion. However, the basic components formed during the explosion of a gaseous mixture are constant and their percentage content in the mixture varies within narrow limits: carbon monoxide 50-60 percent, carbon dioxide 20-25 percent, and nitro gases 15-20 percent (Sapozhnikov, Smol'nikov).

In the light of the given literature data, poisoning by explosion gases such as those described above is still more obvious, especially as it includes symptoms of poisoning by both carbon monoxide and nitrogen oxides. Intoxication by carbon monoxide is shown by the clinical symptoms given above, which were present in varying degrees in all the victims (headache, giddiness, nausea, vomiting) and also by the fact that carboxyhemoglobin was detected in the blood of Victim S.

Intoxication by nitro gases is shown by the emphysema phenomena observed in two patients (in particular, by the characteristic color of the sputum).

Thus, the phenomena described are in full accordance with the symptom complexes of intoxications by carbon monoxide and nitro gases. It should also be noted that among the disorders which we observed it was not possible to detect a syndrome characteristic of intoxication by carbon dioxide, to which some authors (in particular, Ekher [sic] and Isten) attach great importance in the development of disorders which occur due to the effect of explosion gases.

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